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Dispositional Optimism and the Mechanisms by Which It Predicts Slower Disease Progression in HIV: Proactive Behavior, Avoidant Coping, and Depression

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Abstract

The issue of whether optimism may prospectively protect against disease progression is one that has generated much interest, with mixed results in the literature. The purpose of this study was to determine whether dispositional optimism predicts slower disease progression in HIV. Two indicators of disease progression, CD4 counts and viral load, were assessed over 2 years in a diverse group (men, women, White, African American, Hispanic) of 177 people with HIV in the midrange of disease at entry to the study. Optimism predicted slower disease progression (less decrease in CD4 and less increase in viral load) controlling for baseline CD4 and viral load, antiretroviral treatment, gender, race, education, and drug use. Those low on optimism (25th percentile) lost CD4 cells at a rate 1.55 times faster than those high on optimism (75th percentile). Optimists had higher proactive behavior, less avoidant coping, and less depression: These variables mediated the linear optimism–disease progression relationship. Thus, optimists may reap health benefits partly through behavioral (proactive behavior), cognitive (avoidant coping), and affective (depression) pathways. Implications, limitations, and interpretations are discussed.

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Keywords

optimism; coping; HIV/AIDS; HIV disease progression; proactive; depression

After years of examining the possible impact of negative factors such as stressful life events, depression, and hostility on disease progression and quality of life after the diagnosis of devastating illnesses, behavioral medicine is now turning its attention to the possible protective effects of positive psychological factors such as meaning, control, and optimism (reviewed in Chesney et al., 2005; Taylor et al., 2000). One of the most intriguing of these is optimism. There is the popular belief that maintaining hope or optimism may influence survival. Many doctors encourage their patients to develop a positive attitude believing it may be helpful to patients (Schofield et al., 2004). However, there have only been a few studies scientifically examining this.

One study gaining widespread attention (Schofield et al., 2004) recently found that dispositional optimism did not predict progression-free survival in patients with lung cancer and concluded that "encouraging patients to 'be positive' may only add to the burden of having cancer while providing little benefit" (p. 1276). Schulz, Bookwala, Knapp, Scheier, and Williamson (1996) found that, although dispositional optimism did not predict survival in patients with recurrent or metastasized cancer receiving palliative radiation treatment, pessimistic life orientation was a risk factor for mortality but only in younger (age < 59) patients. In contrast, dispositional optimism predicted survival over 1 year in head and neck cancer patients (Allison, Guichard, Fung, & Gilain, 2003). Several studies not restricted to cancer patients found that dispositional optimism is related to better health outcomes including better recovery and less rehospitalization from coronary bypass surgery (Scheier et al., 1989; Scheier et al., 1999) and lower all cause and cardiovascular mortality (Giltay, Geleijnse, Zitman, Hoeskstra, & Evert, 2004). In addition, one study found that a "cognitive adaptation index," which included dispositional optimism, situational-specific optimism, self-esteem, and mastery, predicted fewer new coronary events after Percutaneous Transluminal Coronary Angioplasty (PTCA) (Helgeson & Fritz, 1999).

Although most of the studies noted previously focused on dispositional optimism (generalized positive expectancies regarding future outcomes; Scheier & Carver, 1985), another approach, optimistic attributional style (Peterson & Seligman, 1984), has also been related to better health across the lifespan (Peterson & Seligman, 1987; Peterson, Seligman, & Vaillant, 1988). An optimistic explanatory style is one that uses attributions for negative events that are external, unstable, and specific rather than internal, stable, and global. This latter type of optimism, however, is only modestly correlated with dispositional optimism. Although the focus of this article is on dispositional optimism, studies of attributional optimism are also noted.

Few studies have been undertaken to determine whether or not optimism prospectively predicts disease progression in HIV. Tomakowsky, Lumley, Markowitz, and Frank (2001) reported unclear results: Dispositional optimism was unrelated to CD4 counts over 2 years, but an optimistic explanatory style was a "substantial" predictor of greater decline in CD4 counts over 2 years. Another study (Reed, Kemeny, Taylor, Wang, & Visscher, 1994) found no association between dispositional optimism and HIV progression. In contrast, optimistic outlook, including anticipating future activities, was related to lower mortality during follow-up in a group of hemophiliacs with HIV (Blomkvist et al., 1994). Dispositional optimism was related to less distress and better cellular immunologic control over Epstein-Barr virus and human herpes virus-6 in the months following Hurricane Andrew in people with HIV (Cruess et al., 2000). An inversely related construct, HIV-specific negative

expectancy has been related to earlier symptom onset in bereaved men with HIV (Reed, Kemeny, Taylor, & Visscher, 1999). The most recent study (Milam, Richardson, Marks, Kemper, & McCutchan, 2004) found that dispositional optimism was not linearly related to CD4 or viral load over 18 months; however, optimism had a curvilinear relation with CD4 counts at follow-up (such that moderate levels of optimism at baseline predicted the highest CD4 counts later), and pessimism had a linear relation with higher viral load at follow-up.

In addition to Milam et al.'s (2004) curvilinear findings, the possibility that optimism may not always be beneficial has been suggested by studies of healthy people. For example, Segerstrom (2001) found that optimistic law students had poorer immune status (i.e., lower CD4 cells) when facing academic–social goal conflict. Sieber et al. (1992) found that optimists had a greater decrease in natural killer cell cytotoxicity when exposed to uncontrollable stress (noise). Similarly, F. Cohen et al. (1999) found that optimists showed greater immune decrements than pessimists when stress was maintained at high levels over 3 months. As Segerstrom et al. (2003) note "Optimists' positive moods and confidence" may be "beneficial when coping efforts are effective" but could "lead to greater disappointment and distress when efforts are thwarted or unsuccessful" (p. 1616). These findings suggest that a comprehensive examination of the relation between optimism and immunity should include a consideration of both linear and curvilinear relations.

As Taylor, Kemeny, Reed, Bower, and Gruenewald (2000) note, HIV provides a very useful model for examining psychosocial influences partly because one has meaningful biological markers (CD4 and viral load) to follow and one can control for confounding variables such as age, drug use, sleep, medication use, and so forth. A concern Taylor et al. (2000) raised was whether the discovery of protease inhibitors might limit the use of HIV as a disease model. Most of the studies examining optimism and HIV have been undertaken in samples of men before the advent of powerful medications (protease inhibitors) for HIV. Because of this a statistical modeling procedure, hierarchical linear modeling (HLM; Bryk & Raudenbush, 2002) was selected that allows for the control of time varying covariates (i.e., antiretroviral medication at every time point) and predicts to slope rather than a single point. Thus, the first purpose of this article was to determine whether dispositional optimism would predict disease progression in HIV (both CD4 cell count and HIV-1 viral load) in a diverse sample (e.g., men and women, different ethnic groups) in the era of powerful antiretroviral medications.

A second purpose of this article was to explore mediators of the optimism–disease progression relation. Affective, cognitive, and behavioral pathways were considered. Several studies have shown, for example, that optimism prospectively predicts a resistance to the development of depression and better emotional recovery in various medical populations including women undergoing breast cancer surgery (Carver et al., 1993; Schou et al., 2004) and patients undergoing treatment for metastatic melanoma and renal cell carcinoma (L. Cohen et al., 2001). Optimism has also been related cross-sectionally to less distress in men with HIV (Taylor et al., 1992). Thus, one plausible mechanism by which optimism could impact on disease progression is by resistance to negative emotional states such as depression. Depression, in turn, has been shown to predict disease progression in a number of studies see (Leserman, 2003, for a review).

A second pathway by which optimism could impact CD4, viral load change, or both is through coping. Optimism has been positively related to active coping efforts (Aspinwall & Taylor, 1992; Taylor et al., 1992). These efforts may in turn mitigate the effect of the stressor both affectively and physiologically. Furthermore, optimists persist in goal attainment even in the face of obstacles (Carver & Scheier, 1998). As noted in Segerstrom, Taylor, Kemeny, and Fahey (1998), dispositional optimists make less use of avoidant coping

strategies such as denial and giving up, which have been associated with negative affect (Taylor et al., 1992) and the development of symptoms and death in HIV (Ironson et al., 1994).

Health behaviors could also provide a connection between optimism and health (Mulkana & Hailey, 2001). In men at risk for HIV, Taylor et al. (1992) showed that men who were unrealistically optimistic about their ability to stave off the HIV virus nonetheless practiced better health habits. However, they found that there was no relation of optimism to risk-related sexual behavior. In contrast, Holmes and Pace (2002) found that optimistic beliefs about prognosis were significantly related to both poor medication adherence and risky sex. Finally, drug use and sleep could also be plausible pathways by which optimism could affect immune function (Kiecolt-Glaser & Glaser, 1988) and disease progression and were examined as potential mediators.

Thus, this article seeks to examine whether dispositional optimism predicts HIV disease progression and what the mediators of this relation might be. It extends previous literature by having a more diverse sample (30% women, 70% men), predicting to both CD4 and viral load, being undertaken in an era when powerful medications are available, controlling for medications at every time point, and examining possible mechanisms. Finally, both linear and nonlinear prediction are considered.

Methods

Participants

Inclusion criteria—Participants were HIV positive with CD4 counts between 150 and 500 at entry to the study (chosen so they were in the midrange of illness when one starts to get symptoms).

Exclusion criteria—Participants were excluded if they had ever had an AIDS-defining symptom (Category C symptoms such as Kaposi's sarcoma, *Pneumocystis carinii* pneumonia, etc.), had ever had CD4 cell count below 75, were younger than 18 years, had another life-threatening illness (e.g., cancer), were taking medications thought to affect stress hormones (e.g., steroids, propranolol), were actively psychotic or suicidal, were having current alcohol or drug dependence (based on the psychotic screen and alcohol and drug use modules from the Structured Clinical Interview Diagnosis for the American Psychiatric Association *Diagnostic and Statistical Manual of Mental Disorders*, 3rd ed., rev.), had less than an eighth-grade education in English, or had dementia.

Design

This is a longitudinal study in which participants were seen at five time points (every 6 months for 2 years). Accrual lasted 2.5 years. The time period was between 1997 and 2002.

Procedures

At the initial visit, we obtained informed consent and participants were given a questionnaire packet (described in the measures section). They also underwent an interview, which assessed background information, current life stress, questions about the development of new symptoms, medication, and an assessment of exclusion variables (e.g., drug and alcohol dependence, psychotic screen, and cognitive status as noted previously). They also underwent a blood draw for CD4 and viral load and a brief physical examination to check for the development of Category C symptoms. Blood draws were conducted in the morning to control for diurnal variation. Participants were thanked for their time and paid $50. Similar assessment procedures were followed at each of the 6-month follow-up time points.

Measures

Demographics—Demographics included age, gender (1 = male, 0 = female), ethnicity (1 = White, 0 = other), sexual orientation (1 = heterosexual, 0 = gay/bisexual), and socioeconomic status (SES; education, employment, income). Education was used to represent SES in the HLM models, because it cannot be affected by disease whereas employment and income may be. Education was assessed as less than high school; high school or more, but less than college degree; or college graduate or more.

Background medical information—Background medical information includes CD4 count, viral load, prescribed antiretroviral medications, route of infection, and additional (possibly control, possibly predictor) variables that could affect the course of HIV (including past history of sexually transmitted diseases, sleep, alcohol and drug use, adherence to medication, exercise). Antiretrovirals were dummy coded at each time point reflecting three levels: no medication, combination therapy, or highly active antiretroviral treatment (HAART, which during this study period was combination therapy including a protease inhibitor or Ziagen [abacavir], Sustiva [favirenz], or Viread [tenofovir]).

Disease progression markers—Peripheral blood samples were collected from all participants in ethylenediaminetetraacetic acid (EDTA) tubes (Vacutainer EDTA, Becton-Dickenson, Rytherford, NJ). The percentage of CD4 lymphocytes (CD3+, CD4+) was determined by whole-blood four-color direct immunofluorescence using a Coulter XL-MCL flow cytometer (Coulter Epics XL-MCL, Miami, FL) in line with the method described by Fletcher, Maher, Patarca, and Klimas (2000). Lymphocyte counts were determined using a Beckman/Coulter Hmx hematology analyzer (Beckman/Coulter, Hialeah, FL). The CD4 count was the product of the lymphocyte count and the percentage of lymphocytes positive for both CD4+ and CD3+ markers. Viral load was determined on EDTA plasma using an *in vitro* reverse transcriptase polymerase chain reaction assay (Amplicor, Roche Laboratories, Indianapolis, IN). Results were expressed as HIV-1 ribonucleic acid (RNA) copies per milliliter. A log transformation was used to normalize the distribution.

Psychosocial measures—Optimism was measured by a composite of the Life Orientation Test (LOT; Scheier & Carver, 1985) and the LOT-R (Scheier, Carver, & Bridges, 1994), which assess dispositional optimism or the extent to which one generally anticipates positive outcomes (e.g., I always look on the bright side of things). Participants were asked the degree to which they agreed with each of nine substantive statements (and one filler, not scored) on a scale from 1 to 4. This scale differs from the often used 5-point scale because it does not have a neutral (neither agree nor disagree) option. Higher scores represent higher optimism. Depression was measured by the Beck Depression Inventory (BDI; A. T. Beck, 1967; Beckman & Leber, 1985). Perceived stress was measured by the Perceived Stress Scale (PSS; 10-item version; S. Cohen & Williamson, 1988), which measures “the degree to which situations in one’s life are appraised as stressful.” Coping was measured by the COPE (Carver, Scheier, & Weintraub, 1989). Two subscales were combined to produce an avoidant coping composite (denial and behavioral disengagement). An adaptive coping composite was also constructed (via factor analysis) from five subscales —planning, positive reframing, active coping, acceptance, and emotional support.

Behaviors—Adherence was measured by the proportion of missed doses by self-report on the AIDS Clinical Trial Group (ACTG) adherence measure (Chesney et al., 2000). Drug and alcohol use in the past month were assessed by self-report using a 5-point Likert scale (no use, once or twice, once a week, several times a week, or daily) for the following substances: alcohol, cigarettes, marijuana, and cocaine. Sleep and exercise were assessed through self-report asking “How much sleep did you get each night during the past week?”

and the number of hours of physical exercise during the past week. Safe sex was measured by reported use of condoms (for those who were sexually active). Proactive behavior was measured by ratings from interviews. The interviewer asked a variety of questions, the most relevant of which are how the research participant has been dealing with HIV, what he or she is doing to keep healthy, and whether he or she is satisfied with the doctor. Proactive behavior included reports of self-initiated action-oriented behavior. Examples included information seeking, changing a health behavior, seeking medical care, seeking another doctor's opinion, refusing to be a victim or fighting spirit, and seeking mental health and substance abuse counseling. Reliability for this scoring system between two raters (n = 21) scoring written material was r = .86 (K.D. and H.M.K.). A subset of 15 participant interviews were also rated by blind raters (J.G. and J.W.) yielding an r of .66, p = .007; Kendall's τ-b of .53, T = 2.869, p = .004. In only 1 of the 15 participants did the ratings (done on a scale of 0 to 3) differ by more than 1.

Statistical Methods

The main analyses used HLM (Bryk & Raudenbush, 2002) to predict CD4 and viral load change over time. Key features that led to the choice to use this technique for analysis were that it allows one to control for antiretroviral use at each time point, it allows one to predict to slope of change rather than a single endpoint, and it allows one to calculate the expected drop in CD4 cells (or increase in viral load) over a period of time for any given score of a psychological variable. In this data analytic approach, variance in disease progression markers is separated into two levels. Level 1 represents a growth model for each individual capturing within-person change in CD4 and (log) viral load over repeated measurements. Leve l2 represents amodel of interindividual differences in parameters of individual change and uses between-person characteristics such as education and psychological variables to predict change. Thus, systematic variability of slopes and intercepts at Level 1 are modeled by predictors at Level 2.

Covariate Selection

A priori variables controlled for included initial disease status (CD4 and viral load at study entry—controlled through the intercept in the HLM model), prescribed treatment (antiretrovirals as a time-dependent covariate), and time since entry into the study. Other covariates that were considered based on prior HIV literature (Balbin, Ironson, & Solomon, 1999; Ickovics et al., 2001, Leserman et al., 2000) included demographics (race, gender, age, SES; education was used because it is not affected by HIV as income and employment are) and background variables relevant to HIV (sexual orientation, route of infection) or that impact on the immune system (Kiecolt-Glaser & Glaser, 1988; drug and alcohol use in past month, smoking, exercise, sleep). Covariates were tested individually after controlling for antiretrovirals, baseline health status, and time since entry to the study. Those covariates having $p < .10$ were kept in the model and tested simultaneously. Those still significant at $p < .10$ were kept in the resulting model. Covariates that had been excluded were then retested with all other significant covariates to determine if they should be reentered. After determination of covariates, the final models added optimism as a Level 2 predictor.

Of additional note, recognizing the possibility that health status might affect optimism suggests that optimism should perhaps be controlled for in the intercept at Level 2. However, the correlation between initial CD4 count and optimism was not significant ($r = -.02$) nor was the correlation between optimism and initial viral load (r = .06).

All continuous variables in the model were centered and all categorical variables were coded with zero as the lowest level. HLM parameter estimates account for missingness on the

outcome variables by using full maximum likelihood estimation. The log of viral load was used rather than absolute amount as the variable was skewed.

The two level equations including significant covariates are detailed in Table 1 (with optimism in the model) and summarized as follows.

For CD4

Level 1: $Yti = \beta_{0i} + \beta_{1i}(\text{months since baseline})_{ti} + \beta_{2i}(\text{antiretroviral 1})_{ti} + \beta_{3i}(\text{antiretroviral 2})_{ti} + \beta_{4i}(\text{antiretroviral 1} \times \text{time})_{ti} + \beta_{5i}(\text{antiretroviral 2} \times \text{time})_{ti} + e_{ti}$

where Yti is the CD4 count for participant i at time point t,

β_{0i} is CD4 at entry to the study for the ith participant,

β_{1i} is the slope representing linear change in CD4 over time for participant i,

β_{2i}, β_{3i}, β_{4i}, β_{5i} are the slopes for the antiretrovirals (two variables dummy coded representing the three levels of medication—none, combination therapy, or HAART) and the interaction of antiretrovirals and months since baseline,

e_{ti} is a residual term for participant i at time t.

To examine individual differences in Level 1 change parameters, the Level 2 equations are as follows:

β_{01} (intercept) $= \gamma_{00} + \gamma_{01}$ (gender)$_i + \gamma_{02}$ (ethnicity-white/not)$_i + u_0$

β_{1i} (slope) $= \gamma_{10} + \gamma_{11}(\text{education})_i + \gamma_{12}(\text{marijuana use})_i + \gamma_{13}(\text{optimism}) + u_1$

$\beta_{2i,3i} = \gamma_{20,30}$(antiretroviral 1 or 2),

$\beta_{4i,5i} = \gamma_{40,50}$(antiretroviral 1 or 2 × time)

where γ_{00} represents the group average initial CD4,

γ_{10} represents the average linear change in CD4,

γ_{20} and γ_{30} represent the average effect on level of CD4 across patients of being on antiretroviral 1 or 2 (two dummy coded variables representing three levels of medication—none, combination therapy, or HAART) on level of CD4,

γ_{40} and γ_{50} represent the average effect across patients of antiretroviral 1 and antiretroviral 2 on change in CD4 over time,

γ_{13} represents the effect of individual differences on CD4 slope (γ_{10}) attributable to putative psychological variables beyond the effect of other covariates in the model.

For viral load

Level 1: $Yti = \beta_{01} + \beta_{1i}(\text{months since baseline})_{ti} + \beta_{2i}(\text{antiretroviral 1})_{ti} + \beta_{3i}(\text{antiretroviral 2})_{ti} + e_{ti}$

To examine individual differences in Level 1 change parameters, the Level 2 equations are as follows:

β_{01}(intercept) $= \gamma_{00} + \gamma_{01}(\text{age})_i + \gamma_{02}(\text{gender})_i + \gamma_{03}(\text{cocaine use})_i + u_0$

β_{1i} (slope) $= \gamma_{10} + \gamma_{11}(\text{education})_i + \gamma_{12}(\text{sexual orientation})_i + \gamma_{13}(\text{optimism}) + u_1$

$\beta_{2i,\,3i} = \gamma_{20}, \gamma_{30}$ (antiretroviral 1 or 2)

where Yti is the viral load (log) for participant i at time point t,

β_{0i} is viral load (log) at baseline for the *i*th participant,

β_{1i} is the slope representing linear change in viral load over time for participant *i*,

e_{ti} is the residual or random error term associated with time point *t* for participant *I*,

u represents random error associated with estimation of the *B* coefficients,

β_{2i} and β_{3i} are the average effects across patients being on antiretroviral 1 and antiretroviral 2, respectively.

Results

Description of the Sample

Our sample (*N* = 177) was diverse: 30% female, 70% male; 31% white, 36% African American, 28% Hispanic, and 5% other. The average age was 37.5, and the SES was low to moderate (62% make less than $10,000/year; 27% are college graduates). Thirty-six percent had a history of alcohol abuse, 31% had a history of cocaine abuse, and 24% had a history of *Cannabis* abuse. Fifty-one percent obtained HIV from gay or bisexual sex, 38% from heterosexual sex, 5% from intravenous drug use, and the rest from multiple or other sources. At entry to the study, the average CD4 was 296.7 (*SD* =102.45), and average viral load was 44,861. Seventy-seven percent were on antiretroviral medications (56.5% were on HAART). Complete details of the demographics and medical information for the sample are given in Ironson et al. (2005).

Optimism and Disease Progression

Table 1a presents the HLM model with significant covariates for the prediction of CD4 slope over 2 years, and Table 1b presents the HLM model for the prediction of viral load (log). As can be seen, there was a significant decrease in CD4 over time, and a significant increase in viral load (log). Controlling for significant covariates, optimism measured at baseline predicts the change in CD4 (slope) over 2 years: $\gamma_{13} = 0.19$, $t = 2.08$, $df = 173$, $p = .04$. Although the sample as a whole loses 3.13 CD4 cells per month,[1] optimists gain 0.19 CD4 cells per month for every one point on the LOT. For example, the sample as a whole loses $3.13 \times 12 = 37.56$ CD4 cells per year. A high optimist who is 3.8 points above the mean on the LOT (at the 75th percentile) would gain $0.19 \times 3.8 = .72$ CD4 cells per month or 8.67 CD4 cells per year above the −37.56 for the sample as a whole for a total loss of only 28.89 CD4 cells a year. A low optimist, on the other hand (at the 25th percentile) is 3.2 points below the mean and would lose an additional $-0.19 \times 3.2 = -0.61$ CD4 cells per month or 7.32 CD4 cells per year above the −37.56 for the sample as a whole, which translates into a decline of 44.88 CD4 cells per year. The decline ratio for low versus high optimists may then be calculated to be 44.88/28.89 or 1.55. Thus, low optimists lose 55% more CD4 cells per year than high optimists. Optimism also predicts the change in viral load (log) over time: $\gamma^{13} = -0.001$, $t = -2.007$, $df = 173$, $p = .04$. In this case, high optimists have a slower increase in viral load (log) over time compared to low optimists.

Correlates of Optimism

The first step in exploring possible mediators of the relation between optimism and disease progression was to determine which of our hypothesized mediators were significantly correlated with optimism. Three types of variables were considered: (a) mood/affect (BDI, PSS); (b) coping (avoidant, adaptive); and (c) behaviors, which included safe sex (condom

[1]The average decline in CD4 cells of 3.13 per month is estimated using γ_{13} based on average scores on the continuous variables and zero on the categorical variables, controlling for the covariates in the model.

use if sexually active), adherence to medications, drug use (marijuana, cocaine, alcohol, cigarette smoking), exercise, sleep, and proactive behavior. Optimism was significantly correlated with these psychosocial variables: depression ($r = -.66$, $p < .01$), perceived stress ($r = -.62$, $p < .01$), avoidant coping ($r = -.29$, $p < .01$), and adaptive coping ($r = .44$, $p < .01$). In addition, optimism was significantly correlated with these behaviors: adherence ($r = .18$, $p < .05$), cocaine use ($r = -.28$, $p < .01$), cigarette smoking ($r = -.22$, $p < .01$), exercise ($r = .17$, $p < .05$), and proactive behavior ($r = .26$, $p < .01$). Optimism was not significantly related to practicing safe sex ($r = .06$), alcohol use ($r = .01$), or sleep ($r = .12$).

Mediator Analysis

In addition to being related to optimism, several other criteria as outlined in Baron and Kenny (1986) need to be met. That is, the putative mediator must also predict the outcome variable (CD4 or viral load change), and when both the mediator and optimism are in the equation together, the mediator remains significant and optimism is no longer significant. For partial mediation, optimism is still significant, but the path from optimism to disease progression is reduced in absolute size—optimism explains less of the variance in the dependent variable/disease progression—when the putative mediator is controlled (i.e., added into the equation) compared to when optimism was a predictor without the mediator. Table 2 has the previously mentioned analyses for testing for prediction and mediation (i.e., Table 2 includes only variables correlating with optimism, as noted in the previous paragraph, that also predicted either CD4 or viral load change). The last column, the Sobel test (Sobel, 1982), tests whether a mediator carries the influence of an Independent Variable (IV) to a Dependent Variable (DV) (Preacher & Leonardelli, 2003) or, more precisely, whether the indirect effect of the IV on the DV via the mediator is significantly different from zero.

Mediators of optimism to CD4 change—As can be seen from Table 2, depression was the first variable to qualify as a mediator as it predicts CD4 change, and when both optimism and depression are in the model, depression continues to predict but optimism does not. (Note that examined the other way around, optimism was not a mediator of the depression–disease progression relation.) Finally, the Sobel test was significant. Proactive behavior also mediated the relationship between optimism and CD4 change. Finally, avoidant coping was not a mediator.

One might argue that true mediation should involve measurement of the mediator at a later time point. Because of this, the mediator analyses were repeated with measurement of the mediators at Time 2 (6 months after baseline assessment). These analyses are presented in Table 3 and illustrate that the findings hold for depression and proactive behavior. Furthermore, avoidant coping emerged as a significant mediator when it was measured at Time 2.

Mediators of optimism to viral load change—Similarly, depression qualifies as a mediator because it predicts viral load (log) change, and when both optimism and depression are in the model, depression continues to predict but optimism does not. As before, the Sobel test is significant. Avoidant coping also mediated the relation between optimism and viral load (log) change. Follow-up analyses examining denial and behavioral disengagement separately suggested that behavioral disengagement was the subscale that was driving this effect more powerfully. Proactive coping did not mediate the optimism–viral load relation. As before, one might argue that true mediation should involve measurement of the mediator at a later time point. Because of this, the mediator analyses were repeated with measurement of the mediators at Time 2. These analyses are presented in Table 3 and illustrate that the

findings hold for depression. However, they did not hold for avoidant coping or proactive behavior.

Exploration of curvilinear relations—Based on the findings of Milam et al. (2004), further analyses were undertaken to determine whether there was a curvilinear relation between optimism and either CD4 or viral load. When the quadratic term for optimism was entered into the model with the other covariates plus linear optimism in the model, there were nonsignificant trends for the quadratic optimism term to predict viral load (t^{172} = −1.86, p = .06) and CD4 (t_{172} = 1.59, p = .11).

Discussion

Dispositional optimism predicts two markers of HIV disease progression in an era when very potent antiretrovirals are available and in a very diverse sample. Further analyses suggest that optimism may be related to disease progression through several mediators: more proactive behavior, less depression, and less avoidant coping. Thus, optimists may reap health benefits partly through behavioral (proactive behavior), cognitive (avoidant coping), and affective (depression) pathways. In other words, maintaining a positive out look may serve to keep people more behaviorally engaged and less avoidant (i.e., better able to face the facts) and may confer resistance to depression.

Correlates and Mediators

Consistent with other studies, we found significant relations between optimism and affective variables, such as depression, and with avoidant and adaptive coping, but no relation between optimism and sexual behavior was observed. Less studied are relations between optimism and adherence to medications, drug use (marijuana, cocaine, alcohol, cigarette smoking), exercise, sleep, and proactive behavior. In our sample, optimism was significantly correlated with many of these behaviors: adherence, exercise, cocaine use (negatively), and cigarette smoking (negatively), but none of these met the criteria for mediation. Finally, one new variable that optimism was both related to and met the criteria for a mediator of the optimism–disease progression relation is proactive behavior. Thus, this study extends the literature by showing not only associations but also that proactive behavior, depression, and avoidant coping are mediators of the optimism–disease progression relation.

Although the optimism–proactive behavior–disease progression relation is relatively new, it is consistent with the literature. Carver and Scheier (1998) suggest that optimists are more likely to persist toward goal attainment even in the face of obstacles or stressors. In one study, optimists remained more persistent on a vigilance task than pessimists even when given false-negative feedback (Helton, Dember, Warm, & Matthews, 1999). The literature reviewed by Segerstrom, Castañeda, and Spencer (2003) suggest that optimists may have an easier time persisting with controllable rather than uncontrollable stressors, situations in which there is not goal conflict, and situations in which coping efforts make a difference. Since the advent of powerful medications for HIV, the management of this illness has become more controllable, and coping efforts can make a difference. In addition, Scheier and Carver (1987) suggest that pessimists are more emotion focused under stress than optimists, which would also be consistent with a prediction that optimists would remain more problem focused and perhaps more proactive. In the long run, this disposition could lead to better health management. Thus, optimists may be more action oriented despite the stresses in life. This interpretation would also be consistent with learned helplessness theory such that optimists may have had more responsive early learning environments and have learned that persistence and proactive behavior are more likely to be rewarded (Peterson & Seligman, 1987; Peterson et al., 1988).

Depression was also identified as a mediator of the relation between optimism and disease progression. Thus, optimism appears to act at least partially via depression. Other investigators have raised the question of whether measures of depression and optimism are distinct from each other (Smith et al., 1989) or may both reflect underlying negative affectivity or neuroticism. Although this is one possible interpretation of the high correlation between optimism and depression found in this ($r = -.66$) and other studies, another interpretation is that depression is a conceptually distinct but highly correlated construct. The measure of depression used in this study was the BDI, which assesses several domains: cognitive, affective, and somatic symptoms across 21 items (A. Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961). The only item that can be directly thought to be measuring the same construct (inversely) as optimism is the hopelessness item (e.g., I feel discouraged about the future). In addition, the BDI assesses depressive symptoms over the previous week, whereas the time frame on the LOT is not specified but rather refers to a generalized "way of looking at life." Related to this, depression can be conceptualized as a state measure thought to be more amenable to change (Vos et al., 2004) as compared with dispositional optimism, which is thought to be more of a trait measure. Second, one would expect depression and optimism to be highly correlated because the literature on depression suggests that negative, nonoptimistic thinking is part of the problem that depressed people have (Beck, 1967). Similarly, optimism may confer a resistance to depression. Third, the two variables performed differently in the mediator analysis: Optimism was not a mediator of the depression–disease progression relation (see Table 2 and Table 3). In conclusion, there may well be areas of shared variance among optimism, trait neuroticism, and depression; however, depression, assessed via the BDI, measures state depression, and our data suggest one legitimate possibility is to view depression as a mediator of the optimism–disease progression relation. Although our data are supportive of this interpretation, to establish a true mediator causality rather than prospective correlational analysis (even using an intervening time point) needs to be demonstrated.

Reconciliation With Other HIV Literature

Our study findings are consistent with the Blomkvist et al. (1994) study, in which optimistic outlook and anticipating future activities was related to lower mortality during follow-up in a group of hemophiliacs. However, the findings of this study contrast with the Tomakowsky et al. (2001) study that did not find a relationship between dispositional optimism and CD4 change, the Milam et al. (2004) study that did not find a linear relationship between optimism and CD4, and the Reed et al. (1994) study that did not find a relationship to survival time. There are several possible reasons for these discrepancies: Most importantly, in all three negative studies the linear contribution of optimism was tested after controlling for important psychological variables such as negative affectivity (in Tomakowsky et al.), depression (in Milam et al.), and coping (in Reed et al.) that were conceptualized in this study as potential mediators. (Note that in our study, see Table 2, the linear prediction from optimism is no longer predictive if one controls for depression, which is a variable related to negative affectivity or if one controls for avoidant coping.) Second, there were differences in other covariates, for example, the Tomakowsky et al. study controlled only for whether or not the patient was taking zidovudine (likely because that was the only relevant medication at the time), and the Milam et al. study included only patients on Antiretroviral Therapy (ART), whereas this study controlled more specifically for type of medication (none, combination therapy, HAART) and did so at every time point. Our study also controlled for recreational drug use (marijuana and cocaine). In addition, our study used the same laboratory for all CD4 and viral load measurements, whereas the Milam et al. study used medical records, which may have introduced method variance. Fourth, there were differences in the samples: Two studies followed only men and one study followed predominantly men (Milam et al., 88% men), whereas ours followed a diverse group of men

and women. In addition, the sample sizes differed (n = 47 in Tomakowsky et al., n = 78 in the Reed et al. study, n = 177 in our study, and n = 412 in the Milam et al. study). Another important difference is that our sample was restricted to those in the midrange of HIV at study entry (our sample was restricted to those with CD4 counts between 150 and 500 at baseline) where we hypothesized that the impact of psychological variables would not be overwhelmed by advanced biological deterioration. Finally, in view of these discrepant results (which may in fact, not be all that discrepant as noted previously), more studies need to be conducted to reconcile these results.

Our study also contrasts with the study of lung cancer patients in which optimism did not predict disease course (Schofield et al., 2004) and in patients with metastasized and recurrent cancer (Schulz et al., 1996). One possible reason for a discrepancy is that certain diseases may be more amenable to psychological influences than others. In addition, as noted previously, psychological factors may play a more prominent role at certain stages of a disease, that is, before the biological deterioration is overwhelming. If one's model hypothesizes that psychological variables impact on disease course through the immune and endocrine systems, then cancers that are immunogenic (e.g., melanoma) or virally mediated (e.g., cervical cancer) would be more likely illnesses to find a psychological effect on disease course. Similarly, because the immune system is involved in HIV, and HIV is a virus, there are plausible biological pathways by which psychological factors such as optimism could have an impact.

Curvilinear Associations

Both our study and the Milam et al. (2004) study tested the curvilinear association between optimism and disease progression. We did not find a significant curvilinear relation between optimism and disease progression markers although there was a nonsignificant trend for viral load. Although this result is not definitive, the finding of trends suggests that further exploration of curvilinear models is warranted. It should be noted that the Milam et al. study had a larger sample size, and thus more power, and used different covariates. Most notably the Milam et al. model controlled for depression, which we did not do; we controlled for drugs and three levels of antiretroviral medications changing at every time point.

Limitations and Future Directions

It should be noted that this study only measured dispositional optimism (Scheier & Carver, 1985) and not attributional optimism. As noted, dispositional optimism focuses on generalized positive expectancies regarding future outcomes and is derived from behavioral self-regulation theory. The other approach, formulated by Peterson and Seligman (1984), was derived from the reformulated learned helplessness model and focuses on optimism as an explanatory style. An optimistic explanatory style is one that uses attributions for negative events that are external, unstable, and specific rather than internal, stable, and global.

In addition, this article did not address the chicken or egg question. That is, does optimism lead to continued good health, or does continued good health lead to optimism? This is left for future research and should be answered making a conceptual distinction between dispositional and attributional optimism, because the latter may be more amenable to change. A further question that this study did not address that would be helpful for future studies to address is distinguishing between realistic optimism and unrealistic optimism. Unfortunately, this is hard to define, although not impossible in HIV because there are prognostic disease progression markers.

As suggested previously, further research should identify which diseases and at what point in the course of the disease optimism would be protective of health and immunity. In addition, we did not address the question of the unique effects of optimism versus pessimism. Finally, the results of the curvilinear analyses deserve more attention because the findings showed a nonsignificant trend.

Implications for Clinical Practice

Should doctors tell their patients to maintain a positive outlook? Our results suggest that having a positive, optimistic attitude may be helpful due to its relation with slower disease progression. Mediational analyses suggest that encouraging people to be more proactive, helping them to be less avoidant, and screening and treating them for depression could contribute to some of the protective effects of optimism as well.

On the flip side, unrealistic optimism is likely to be deleterious. As Peterson and Vaidya (2003) note “Optimism is suspect if it leads to pointless persistence” (p. 33). A more useful strategy may be to follow what Seligman (1991) called “flexible optimism” or what Armor and Taylor (1998) call “strategic optimism,” which is a psychological strategy to be exercised when appropriate.

A study suggesting optimism is related to disease progression raises another question: What can be done to raise optimism levels, or, said differently, what can be done to help people maintain hope? Seligman (1991) has written about learned optimism, but how much of this applies or would be useful in a medical situation is unknown. This study has also not addressed the question of whether a doctor-suggested optimistic attitude would make a difference. Furthermore, as noted, the data suggest that one may confer some of the benefits of optimism by encouraging proactive behavior and by preventing depression and avoidant coping. This may be easier and more realistic than trying to impact on dispositional optimism, which may in fact be more of a trait and may have resulted from years of learning and conditioning (and conversely from learned helplessness) or even partially from a genetic predisposition (Schulman, Keith, & Seligman, 1993), neither of which are easy to change. The issue of how much one should encourage patients to be optimistic for other reasons (i.e., quality of life) is beyond the scope of this article but is nonetheless important. Similarly, issues surrounding the fine line of giving enough hope to patients to help them function and enjoy life versus giving them false hope needs to include much more than the statistical findings available from this article.

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Table 1

Table 1a. HLM Model Including Coefficients and Significance Tests for Level 1 and Level 2 Covariates and Optimism in Prediction of CD4 Slope Over 2 Years, Controlling for Retained Covariates

Fixed Effect	Coefficient	Standard Error	*t* ratio	*df*	*p* value
CD4 intercept, β_0					
Average initial CD4, γ_{00}	296.31	17.84	16.61	174	<.001
Gender, γ_{01}	−34.42	17.11	−2.01	174	.044
Ethnicity, γ_{02}	38.62	16.10	2.40	174	.017
CD4 slope (per month), β_1					
Average slope, γ_{10}	−3.13	0.83	−2.46	173	.014
Education, γ_{11}	1.01	0.76	1.34	173	.181
Marijuana use, γ_{12}	−1.07	0.37	−2.90	173	.004
Optimism, γ_{13}	0.19	0.09	2.08	173	.037
Antiretroviral 1 increment, β_2					
Average increment, γ_{20}	48.53	16.75	2.90	718	.004
Antiretrovira1 2 increment β_3					
Average increment γ_{30}	18.00	14.05	1.28	718	.200
Antiretrovira1 1 increment over time, β_4					
Average increment over time, γ_{40}	3.11	1.36	2.28	718	.022
Antiretrovira1 2 increment over time, β_5					
Average increment over time, γ_{50}	3.33	0.94	3.53	718	.001

Table 1b. Prediction of log Viral Load Slope Over 2 Years From Optimism Controlling for Retained Covariates

Fixed Effect	Coefficient	Standard Error	*t* ratio	*df*	*p* value
VL log intercept, β_0					
Intercept, γ_{00}	4.567	0.140	32.67	173	< .001
Age, γ_{01}	−0.014	0.008	−1.85	173	.064
Gender, γ_{02}	−0.288	0.157	−1.84	173	.066
Cocaine use, γ_{03}	0.197	0.094	2.08	173	.037
VL log slope (per month), β_1					

Table 1b. Prediction of log Viral Load Slope Over 2 Years From Optimism Controlling for Retained Covariates

Fixed Effect	Coefficient	Standard Error	*t* ratio	*df*	*p* value
Average slope, γ_{10}	0.013	0.004	2.98	173	.003
Education, γ_{11}	−0.007	0.004	−1.51	173	.130
Sexual orientation, γ_{12}	−0.016	0.006	−2.63	173	.009
Optimism, γ_{13}	−0.001	.001	−2.01	173	.044
Antiretroviral 1 increment, β_2					
Average increment, γ_{20}	−0.997	0.120	−8.29	719	< .001
Antiretroviral 2 increment, β_3					
Average increment, γ_{20}	−1.032	0.112	−9.25	719	< .001

Note. HLM - hierarchical linear modeling.

Note. VL = viral load.

Table 2

Mediator Analysis for Potential Mediators (Measured at Baseline) That Predict CD4 or VL Change

Mediator	Mediator Alone *t*	Mediator Controlling for Optimism *t*	Optimism Controlling for Mediator *t*	Sobel Test |*z*|
Prediction to CD4 slope				
Depression (BDI)	−2.814**	−2.201*	0.352	2.163*
Avoidant coping	−1.855+	−1.418	1.591	1.335
Proactive behavior	3.470**	3.140**	1.636+	2.312*
Prediction to VL(log) slope				
Depression (BDI)	3.358**	2.850**	0.251	2.443*
Avoidant coping	3.097**	2.718**	−1.382	2.171*
Denial	2.245*	2.034*	−1.715+	1.411
Behavioral disengagement	2.881**	2.359*	−1.343	2.122*
Proactive behavior	–0.592	−0.224	−1.835+	0.218

Note. BDI = Beck Depression Inventory; VL = viral load.

* $p < .05$.

** $p < .01$.

+ $p < .10$.

Table 3

Mediator Analysis for Potential Mediators (Measured at Time 2) That Predict CD4 or VL Change

Mediator	Mediator Alone *t*	Mediator Controlling for Optimism *t*	Optimism Controlling for Mediator *t*	Sobel Test |*z*|
Prediction to CD4 slope				
Depression	−3.960**	−2.969**	1.300	2.487*
Avoidant coping	−3.387**	−2.448*	1.340	2.145*
Proactive behavior	3.383**	3.054**	1.675	1.987*
Prediction to VL (log) slope				
Depression	4.055**	3.379**	−0.876	2.714*
Avoidant coping	2.129*	1.212	−1.273	1.169
Proactive behavior	−1.246	−0.916	−1.597	0.865

Note. VL = viral load.

* $p < .05$.

** $p < .01$.